mitral insufficiency; thickening at the base of the mitral cusps; and an increase in the amount of adipose tissue in the pericardium over the right ventricle at the junction of the auricles and ventricles and along the coronary arteries. Microscopically there was fatty infiltration and degeneration in the walls of the right ventricle, and, to a less extent, in those of the left ventricles; and no pathological changes in the coronary arteries. The kidneys showed an acute exudative nephritis with fatty changes; the liver a slight interstitial nephritis, with fatty degeneration of the liver cells, and a moderate venous congestion.

Careful sectioning of the bundle of His, compared with similar sections from a normal heart, showed that the bundle was infiltrated with fat, and had undergone fatty degeneration to such an extent that it was atrophied to not more than one-fifth of its normal size. The result of the autopsy in this case, therefore, tends to confirm the later theories in regard to the nature and causation of Adams-Stokes disease.

POSITION OF THE MOTOR AREAS OF THE HUMAN CORTEX.

By Hermon C. Gordinier, M.D.,
PROFESSOR OF PHYSIOLOGY IN THE ALBANY MEDICAL COLLEGE, ALBANY, NEW YORK.

Case I.—*A rapidly growing glioma of the right motor area (precental convolution) occupying the leg centre, the centres for the arm and face being destroyed by softening. Clinically focal Jacksonian attacks, usually beginning in the left leg, and not preceded by a sensory aura, the leg becoming totally, the arm and lower facial muscles partially, paralyzed as to motion but not as to sensation.*

Mrs. M., aged seventy-six years, was seen on August 10, 1900. Her father died of cancer of the stomach, her mother of pneumonia. She lost two brothers—one of pneumonia, the other of la grippe. Three sisters are living and well. There is no history of insanity or nervous diseases in the family.

The present illness began ten weeks ago with a spasm of the whole left side. This spasm began as a jerky motion of the leg, which was followed by similar movements of the arm and face. The spasm was preceded by no signal symptoms, such as numbness, tingling, or formication, nor by headache, nausea, or vertigo. These attacks, numbering from three to twenty daily, have been repeated ever since. They always begin in the leg or foot, extend upward to the thigh and then involve the arm and face. They never have been followed by loss of consciousness. Since the first unilateral convulsion, she has been slightly paralyzed on the whole left side. She never experiences numbness or pain in the extremities. She states that she is perfectly able to appreciate at all times the exact position of her limbs in space.

She is slightly built, weighs 110 pounds, and is 5 feet 4 inches in height. There is no œdema of the extremities or cyanosis. Physical examination shows moderate general arteriosclerosis and left ventricular hypertrophy. The urine is negative.

The ocular movements are normal. There is no constriction of the visual fields. The pupils are equal and react to light and accommodation. The optic disks are normal. The facial muscles appear symmetrical, but the left nasolabial fold is not so accentuated as is that of the opposite side. The tongue is protruded straight and presents no tremor or wasting. The special senses are normal. All movements of the left arm are somewhat limited and the arm is held in a position of slight contracture. The movements of the left lower extremity are very limited and the foot is held in an equinovarus position. The most careful tests fail to bring out the slightest evidence of ataxia. The tactile, temperature, pain, muscular, and stereognostic senses are normal. The patient is able to appreciate weights and recognizes immediately the position of her limbs in space. The left patellar tendon reflex and the Achilles tendon jerk are greatly exaggerated and there is a distinct ankle clonus on the left side. The Babinski phenomenon is marked. Because of the contracted condition of the muscles of the left arm, it is rather difficult to be sure of the deep reflexes; there is, however, a definite triceps tendon reflex present. The superficial reflexes show no particular alteration. The reflexes of the right side are normal. The visceral reflexes are perfectly controlled. Her mental condition is normal. Up to the present she has had no severe headache.

October 1, 1900. The patient's condition has remained unchanged, save for the development of severe headaches located chiefly over the right forehead and extending down the right side of the neck; they are occasionally accompanied by attacks of projectile vomiting. Complete motor paralysis of the left leg exists, together with paresis of the left arm and lower facial muscles of the left side. The tongue deviates toward the left. She still has an occasional Jacksonian attack, sometimes beginning in the leg and at other times in the arm, and involving subsequently the muscles of the face. The pupillary reflexes are normal, and the optic disks show no changes. Cerebration is somewhat slower than normal, but questions are answered correctly. The head is rotated sharply to the right side. The most painstaking examination fails to elicit the slightest sensory defect.

15*th*. The patient has lost control over the sphincters. She is very dull and lethargic. The headache is much more severe. The left arm is capable of slight motion. The leg is perfectly motionless. The deep reflexes are exaggerated and ankle clonus still persists. The head remains rotated sharply to the right and is held rigidly fixed. She has had no unilateral convulsions for a fortnight.

November 3. The patient gradually passed into a coma and died. A postmortem examination limited to the cranium was made this evening. The following are the notes: The vessels of the pia are injected, especially on the right side. The dura and pia are adherent over the upper part of the right Rolandic area, beneath which exists a hard, somewhat oblong, nodular mass, involving the upper part of the ascending frontal convolution, the bases of the first and second frontal gyri (Fig. 1), and the ventral part of the paracentral lobule (Fig. 2), which is also softened. The growth measures 3 cm. from above downward and 3.5 cm. transversely. The part involving the paracentral convolution is 1.5 cm. in its broadest diameter. The growth is absolutely limited to the upper part of the precentral gyrus, the part of the paracentral convolution corresponding to the median surface of the precentral gyrus, and the bases of the superior and middle frontal gyri. The part of the precentral convolution not occupied by the new-growth is softened, and there is an area of softening in the centrum ovale beneath the tumor mass, which extends forward into the prefrontal lobe. The ventricles appear normal. The arteries at the base are atheromatous. The anterior part of the dorsal limb of the internal capsule appears much grayer than the posterior part. Sections through the crura cerebri, pons, and medulla show no macroscopic changes. Sections of the growth show it to be a glioma.

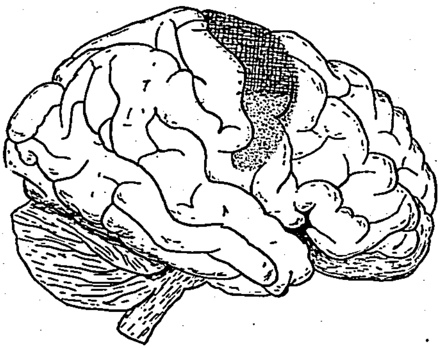

Fig. 1.—Schematic representation of the external surface of the right cerebral hemisphere (Case I). The heavy cross-lines indicate the position occupied by the growth, the dotted lines the area of softening.

The symptoms in this case clearly pointed to an intracranial lesion located in that part of the right motor area, the precentral gyrus, governing the movements of the leg, and subsequently involving the centres for the arm, face, and tongue. This focal diagnosis was supported by the complete absence of sensory symptoms and the presence of typical Jacksonian attacks, beginning in the leg and travelling to the arm and face, to be followed by partial and then by more or less permanent motor paralysis of the left leg, arm, and face, with contracture of the arm and leg, and marked increase of all the deep reflexes.

At first it was difficult to describe just what the pathological diagnosis was, as none of the general symptoms of a growth was present. Later, however, the onset and continuation of severe unilateral headache, together with occasional attacks of projectile vomiting without fever, despite the absence of optic neuritis, indicated that the lesion was probably a tumor.

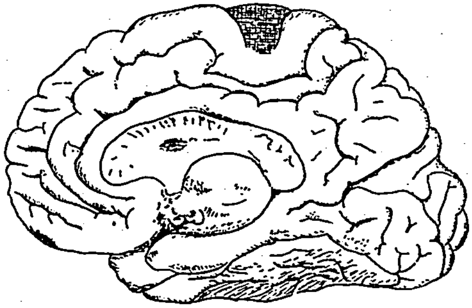

FIG. 2.—The position of the growth on the median surface of the right hemisphere (Case I).

CASE II.—*An abscess of the right motor area (precentral convolution) characterized by unilateral convulsive movements of the muscles of the head and neck toward the left, together with similar movements of the arm and lower part of the face of the same side, without sensory auræ, followed by total paralysis of the left arm and paresis of the face.*

J. E. H., aged fifty-two years, married, a farmer by occupation, entered the Samaritan Hospital, February 17, 1900, under the care of Dr. E. D. Ferguson, who asked me to see him in reference to the possibility of an operation. His father died of rheumatism. His mother is living, but is rheumatic. One brother is living and well. No history of cancer, tuberculosis, insanity, or nervous disease in the family. The patient has had the usual diseases of childhood During the past two years he has had several attacks of indigestion. He denies venereal disease. The present illness began suddenly, January 5, 1900, with a severe unilateral convulsion followed by unconsciousness. The attack began with convulsive movements of the head and eyes toward the left side, then the whole left side of the body became convulsed and the patient lost consciousness. The effects of this attack passed away in a short time and no other occurred until January 31, 1900, the patient appearing perfectly well during the interim. Neither the first nor the second attack was preceded by numbness. The second attack began with convulsive movements of the head and neck toward the left, and almost simultaneously the arm and lower facial muscles of the same side were convulsed. This attack was not followed by loss of consciousness. A half hour later a third attack began—identical in character with the preceding one and without loss of consciousness. On February 14, the fourth convulsive paroxysm occurred, milder in character than the former, accompanied by movements of the muscles of the head, neck, arm, and face of the left side and without loss of consciousness. None of these paroxysms has been preceded by numbness or other sensory disturbances. After the last attack the patient noticed that the movements of his arm were very weak. A gradual failure of memory, particularly for names and dates, has accompanied these attacks. He has never experienced severe headaches, but complains of a constant dull frontal headache. He has noticed no loss of vision. He never vomits. The bowels are constipated; he urinates frequently at night, and has perfect control of the bladder and rectum.

The patient is moderately well built; height, 5 feet 10 inches; weight 160 pounds; healthy; musculature flabby; no cyanosis. Respiration, 16; pulse, 80, regular, full and of fair tension. The veins over the right side of the scalp are prominent. Movements of eyeballs and eyelids are normal. The pupils are midwide and react to light and accommodation. No contraction of the visual fields; vision is normal; the optic disks are normal; no defect of hearing. There is a left-sided, slight facial paralysis of central type, as evidenced by obliteration of the left nasolabial fold and dropping of the angle of the mouth on the same side; there is no rigidity of the neck; the tongue is protruded straight, and no atrophy or fibrillary twitchings were observed. The left arm is held in a position of semiflexion and is partially paralyzed. A slight contracture of the flexor muscles, which is easily overcome, is noticed. All movements of the left leg are normal. The triceps tendon reflex is present on the left side. No wrist tendon reflex or clonus on same side. The umbilical and epigastric reflexes are slightly exaggerated on the left side. The cremasteric is present, but not so active as on the right side. The left patellar reflex is exaggerated, and marked ankle clonus exists. The Babinski phenomenon is present. All reflexes are normal on the right side. The tactile, pain, and temperature senses are normal. The patient is able to recognize at once all objects handled. Cerebration is decidedly slower than normal. When a question is asked, some time elapses before the answer comes. He often errs in names and dates. No aphasic symptoms were present. The patient being right-handed no difficulty in writing was found. No unilateral convulsive seizures have occurred since the patient entered the hospital.

Physical examination of the internal organs is negative. The urine: Specific gravity, 1020; acid; no albumin, no sugar, no pus, no blood, no casts.

A diagnosis of tumor of the right motor area lying upon or just beneath the cortex was made.

February 22, 1900. The patient was anesthetized and the right Rolandic area was exposed after the method of Reid. No pulsation of the brain was noticed beneath the opening. The dura was incised and the vessels of the pia were found injected. No tumor was detected. The opening was then enlarged by a rongeur forceps more especially forward, but despite the large opening nothing was found save a circumscribed area which did not pulsate. This area was explored with fine needles, but meeting no particular resistance it was concluded that the lesion was subcortical and inoperable. The wound was then closed. No hernia cerebri followed. The following day a careful examination was made and the patient's condition was found to be the same as the day before the operation. The optic disks were normal. He did not complain of headache after the operation. A few days later it was noticed that the left arm was almost totally paralyzed. No sensory disturbances were present. No special change occurred in his general condition after the operation until March 12, when he became delirious, lapsed into an unconscious state, and died on the following day.

The postmortem was confined to an examination of the brain. The brain is normal in size and shape and the membranes are free save beneath the defect in the skull, where they are intimately blended, and the dura is adherent to the edges of the overlying (operative) bone defect. There is a slight increase of the cerebrospinal fluid. The right cerebral hemisphere presents a prominence in the region of the central convolutions especially in their upper half. Over this area exists a local collection of subarachnoidean fluid confined by delicate adhesions in the meshes of the pia-arachnoid. After removal of the pia-arachnoid there is found a tumor-like projection which involves the middle third of the anterior central or ascending frontal convolution, and extending forward and upward involves the bases of the superior and middle frontal gyri. This mass produced an evident bulging of the cerebral cortex, arising probably from the centrum semi-ovale, being covered only by a thin shell of cortical tissue half a centimeter in thickness. The mass has an elastic feel as if it contained fluid; it measures 4 cm. in its anteroposterior diameter and 3.5 cm. in its vertical diameter. The exact location of the mass (Figs. 3 and 4) is just beneath the cortex of the middle third of the ascending frontal gyrus, whence it extends forward and upward across the precentral sulcus involving the bases of the first and second frontal convolutions, which gyri are pushed forward by it. The ascending frontal convolution is, from below,

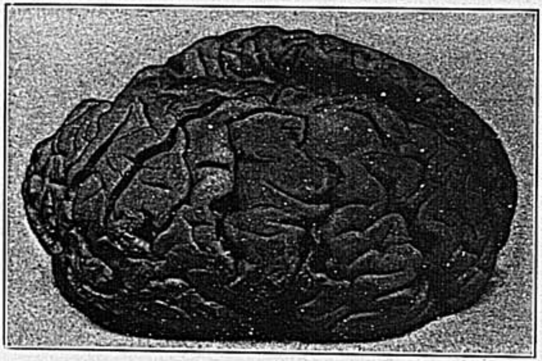

FIG. 3.—The external surface of the cerebral hemispheres, with a tumor-like mass involving the bases of the right first and second frontal convolutions together with the ascending frontal gyrus (Case II).

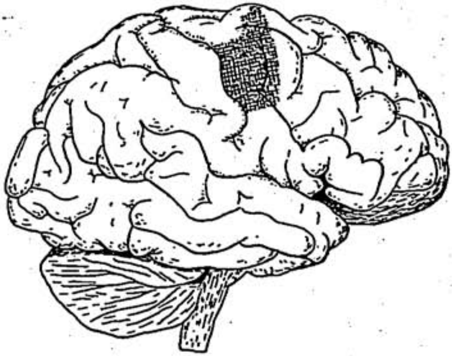

FIG. 4.—The exact position and extent of the abscess as it appeared on the external surface of the right cerebral hemisphere (Case II).

upward, distinctly widened. The cortical portion of the posterior central gyrus is not involved, but the fissure of Rolando in its upper part is narrowed. The superior portion of the ascending frontal, the paracentral lobule, the marginal portion of the superior frontal gyrus, as well as the outer and median surfaces of the superior parietal convolutions are normal.

After carefully hardening the brain in formalin, a series of frontal sections was made, whereupon an abscess was discovered in the right hemisphere, surrounded by a distinct capsule 0.5 cm. in thickness (Fig. 5). The abscess is entirely subcortical and measures, in its anteroposterior diameter, 5 cm.; in its vertical, 2.5 cm.; and in its broadest part, 3.25 cm. The abscess cavity extends forward into the frontal lobe involving the centrum ovale just beneath the cortex of the bases of the superior and middle frontal convolutions. It then broadens out beneath the middle of the anterior central convolution, where it has its greatest development. The superior wall of the capsule is covered by a thin shell of cortex, less than half a centimeter in thickness. The abscess proceeds backward, rapidly diminishing in size and depth, and terminates deep beneath the level of the middle third of the posterior central convolution.

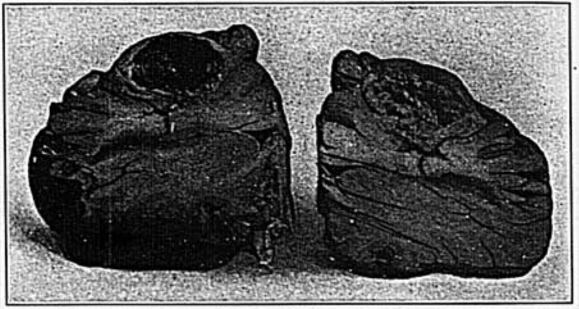

FIG. 5.—Frontal section through the frontal lobe at the precentral fissure and the middle of the ascending frontal gyrus (Case II).

The pus in the abscess cavity was thick, and contained much broken-down tissue. Coverslips taken from it showed a rod-shaped organism resembling one of the "colon" group, which completely decolorized with Gram's method. The identity of this organism could not be determined, as the brain was sectioned after several months of hardening in "formol" solution. The surrounding brain tissue showed only inflammatory changes, evidenced by the presence of numerous dilated vessels, with much small round-cell infiltration, especially in and about the perivascular spaces. The neuroglia elements were not proliferated.

CASE III.—*Recurring clonic spasms of the facial muscles of the right side existing for three years, without aphasia, due to a lesion (scar) of the lower fourth of the left precentral gyrus.*

Mrs. E. F. R., aged fifty years, married. Has had five children, three of whom are living. Her father died at eighty-four years of age, of some obscure disease of the liver; her mother is living and is comparatively well. She suffers from rheumatism. Lost a brother in infancy by cholera infantum. Her husband died of phthisis pulmonalis. She has had all the usual diseases of childhood. She had always enjoyed good health until three years after marriage, when she was infected with syphilis by her husband. Two children were born after her infection, both showing unmistakable signs of congenital syphilis. One died shortly after birth and the other lived until the age of twenty-four years, dying then of tuberculosis. Her present illness began four years ago, when she became nervous and developed severe unilateral headaches, chiefly nocturnal in type, and confined to the left side. The headaches continued about three months, when there occurred a clonic spasm of the facial muscles of the right side. At first these attacks occurred only rarely, but they gradually increased in frequency, so that they recurred every day or two for a period of about three years. They were never preceded

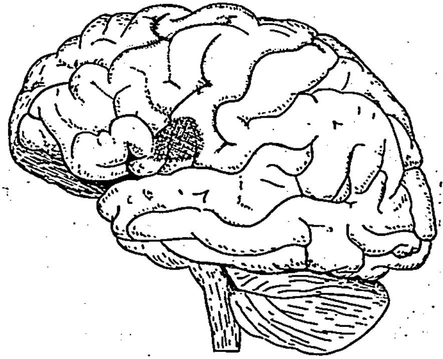

FIG. 6.—The cross-lines indicate the position occupied by the lesion (syphilitic scar) in the left cerebral hemisphere, which excited recurring attacks of right facial monospasm (Case III).

by an aura nor followed by loss of consciousness. She never suffered from numbness, and stated that her vision was excellent.

Examination, October 1, 1900. She is rather short, moderately well built, and weighs 140 pounds. The lungs are negative. The cardiac dulness is increased to the left and the apex is displaced downward and outward. The gait is normal, and all movements of the extremities are perfectly performed. Sensation is unimpaired; no ataxia is present. The patient recognized at once the various objects placed into her hands. There is no mental defect and no aphasia or agraphia is present. The patellar tendon reflexes are lively, otherwise the superficial and deep reflexes showed no deviation from the normal.

The physician was advised to give her potassium iodide with mercurial inunctions. With this treatment there was a gradual improvement; the headaches became less severe, and the facial spasms, though never ceasing, became much less frequent.

I saw her again April 12, 1901, and found her suffering from severe headache. She was also very apathetic and drowsy, with marked failure of memory, but no aphasia. Her renal condition had advanced. The left ventricle is enlarged, the apex being located in the sixth interspace just to the left of the mammillary line. The radial and temporal arteries are thickened; the tension is increased; slight œdema exists over the tibiæ.

The urine: Light amber; specific gravity, 1.012; contains a trace of albumin and granular and hyaline casts.

A paresis of the lower facial muscles of the right side of the face exists. The tongue is protruded straight; there is no difficulty of deglutition. The muscles of the palate functionate normally and there is no loss of the sense of taste or smell. Hearing is perfect. The ocular movements are all preserved. The pupils are equal and react normally to light and accommodation. Vision is perfect. The optic disks are normal. There is no impairment of sensation on either side of the face. There is no tenderness on percussion of the scalp, and no rigidity of the neck.

November 3, 1901, she developed a series of general convulsions, epileptiform in type and died the same evening.

A postmortem, confined to an examination of the brain, was permitted. No change was found in the brain save a localized thickening of the meninges confined to the lowermost part of the left precentral gyrus, the cortex of which had been destroyed by a broken-down gumma and much scar tissue.

In these three cases, which presented during life focal convulsive movements, followed by partial or complete paralysis of motion, not preceded by auræ, and in which the most careful examination failed at any time to disclose the presence of sensory symptoms, the causes were lesions situated ventral to the fissure of Rolando, in the precentral convolution, together with the bases of the superior and middle frontal gyri.

Case I was a glioma destroying the upper third of the precentral gyrus and its paracentral annex, with the bases of the first and second frontal gyri, which at first excited crural spasm and monoplegia followed by paresis of the arm and face, the latter symptoms being probably due to subsequent softening of the remainder of the precentral gyrus.

Case II was an abscess of the middle of the right precentral gyrus and bases of the superior and middle frontal gyri, which caused unilateral convulsive movements of the head and face toward the left, together with similar movements of the left arm and face, followed by complete motor paralyses of the left arm and paresis of the lower facial muscles of the left side.

Case III. The lesion, a broken-down gumma with the formation of scar tissue and consequent atrophy of the cortex of the lower part of the left precentral gyrus, caused for a period of about three years right unilateral facial spasm followed by paresis of the face without motor aphasia; death probably was due to uremia.

In 22 cases, either with autopsies or in which the cortical area was exposed at operations, which I have collected from the literature, presenting during life motor paralysis, preceded or not by Jacksonian attacks and without sensory symptoms, the lesions were invariably located ventral to the fissure of Rolando, involving the precentral convolution together with, in several instances, the bases of one or more of the frontal convolutions.

In 3 of these cases a crural monoplegia was due to a lesion of the superior part of the precentral gyrus and base of the superior frontal convolution of the opposite side. In 6 cases irritative and paralytic symptoms, involving the muscles of the opposite side and unaccompanied by sensory disturbances, were due to circumscribed lesions of the middle portion of the precentral and extreme base of the midfrontal gyrus. In 3 of these cases the lesions were strictly limited to the middle of the precentral gyrus. In 7 of the cases focal convulsive movements of the muscles of the opposite side of the face, accompanied or not by facial paralysis, and without sensory symptoms, were due to limited lesions located in the lower part of the ascending frontal gyrus. In one of these cases motor aphasia was present, the lesion having encroached upon the base of the left third frontal gyrus. In another case pseudobulbar paralysis was present, and was due to lesions symmetrically placed in each hemisphere and limited to the inferior part of the ascending frontal and bases of the middle and inferior frontal gyri.

In 4 cases with partial or complete hemiplegia without sensory loss of "partial or complete hemianesthesia," the lesions were confined to the precentral convolution and paracentral lobule. In 1 case a gumma of the left first frontal convolution, bordering evenly upon the adjacent paracentral convolution, had caused hemiplegia without hemianesthesia.

In another case with frontal lobe symptoms, partial hemiplegia and tonic spasm of the right arm without sensory defect, two tumors were found to explain the symptoms: one at the base of the midfrontal lobule just ventral to the precentral fissure, the other in the ascending frontal gyrus limited in front by the precentral fissure and behind by the fissure of Rolando.

Dieulafoy has recorded a case of paralysis of the right arm and right lower facial muscles, with sensation unimpaired, due to hemorrhagic extravasation the size of a nut in the ascending frontal convolution of the left hemisphere dorsal to the third frontal gyrus. In addition to the above-mentioned cases, I introduce brief abstracts of the following cases taken from Ferrier's article on "Localization" in Allbutt's *System of Medicine.*

Lloyd and Deaver have reported a case of excision of a portion of the hand centre of the right hemisphere, which caused paralysis of the flexors of the left wrist and fingers and left half of the face. Three months after the operation, with the paralysis still existing, the patient was able to feel the slightest touch and was able accurately to recognize weights. He could not distinguish the form of objects because of the inability to move the fingers. Thermal sense was normal.

In a case of von Bergmann's of excision of a portion of the hand centre in the left hemisphere there was not the slightest affection of sensibility in any of its forms.

In Parker and Gotch's case of excision of a portion of the genu of the ascending frontal convolution of the right side, although followed by temporary paralysis of the left hand, no impairment of sensation resulted.

Diller and Buchanan have reported a case with paresis of the right limbs and right side of the face, due to subcortical cyst in the lower part of the ascending frontal convolution. The patient could feel and localize touch in any part of the paretic side and discriminate weights accurately.

Of great value in this connection in support of the doctrine of the definite localization of motor and sensory centres distinct from one another are the cases of partial or complete sensory loss without motor symptoms, due to lesions located dorsal to the fissure of Rolando in the parietal lobe. Redlich, from a study of the position of the lesions in 20 cases, with defects or loss of the muscle sense, believes that we can positively locate a centre for muscle sense in the parietal lobe. Von Monakow, Vetter, Grasset, and Starr have each recorded cases of great defect of the muscle sense without the slightest motor paralysis, with lesions confined to the parietal lobe. In 3 of these cases the central convolutions were found microscopically normal.

Interesting in this connection are the cases of von Monakow, Mills, Walton and Paul, Müller, Spiller, Russel, and Horsley, in which sensory defects of various combinations "tactile temperature, posture, astereognostic pressure, space," etc., unaccompanied by motor paralysis, were due to lesions in the parietal lobe.

It is also worthy of note that in 30 cases studied from the literature with definite motor and sensory paralysis due to cortical disease the lesions invariably involved both central convolutions.

In these days of experimental research this paper would be incomplete without alluding to the very interesting and valuable experiments of Tschermak and Professors Sherrington and Greenbaum, who, operating upon all known species of anthropoid apes, found that with unipolar faradization as the method of cortical excitation the responsive area was limited to the precentral convolution, and to the small coterminous portion of the paracentral lobule on the mesial surface of the hemisphere. Not once in the experience of Professors Sherrington and Greenbaum were movements obtained by excitation of the normal anthropoid postcentral gyrus, nor was experimental ablation of a portion of the same convolution attended by positive interference with motion.

These observations have been further supplemented and largely verified by the valuable experiments upon the cortex of the human brain exposed during operations by Keen, Mills and Frazier, Cushing, Lloyd, and others.

Furthermore, the very recent histological studies of Cajal and Campbell show that a marked difference of construction anatomically exists between the precentral and postcentral gyrus. And of great value in this connection are the studies of Campbell of the changes in the cerebral cortex in cases of amyotrophic lateral sclerosis and tabes dorsales. In the former, the type of a pure motor disease, he found definite changes limited to the precentral convolution, whereas in the latter, a purely sensory one, the pathological changes were limited to the postcentral gyrus.

From a careful study of my own cases and those that I have been able to collect from the literature I have formulated the following two conclusions:

1. There is a determined area in the cerebral cortex presiding over motor functions, and that area is the precentral convolution and its paracentral annex (possibly the bases of the superior and middle frontal gyri may have to be included).

2. The motor and sensory areas of the brain, though interdependent, are, as in the spinal cord, perfectly distinct from one another.

REFERENCES.

Barlow. Brit. Med. Jour., July, 1877.
Bourneville. Soc. de Biol., 1876.
Cestan and Lejonne. Revue Neurol., 1901, p. 846.
Channing and Knowlton. Jour. of Insan., January, 1903, p. 50.
Dreyfous. Bull. d. l. Soc. anat., 1874.

Dieulafoy. Gaz. des hôp., 1868.
Dana. Cutaneous and Muscular Sensations, Jour. Nerv. and Ment. Dis., December, 1894.
Gouguenheim. Topische Diagnostik Nothnagel, p. 408.
Gauche, I. B. Compt. rend. Société de biologie, Paris, 1882.
Hitzig. Archiv. f. Psychiat. u. Nervenkrankh., Band iii, p. 231.
Hervey. Bull. d. l. Soc. anat., 1876, p. 277.
Jackson, H. Brain, 1882–1883, p. 364.
Mahot. Bull. d. l. Soc. anat., 1876, p. 734.
Von Monakow. Nothnagel's Specielle Pathologie und Therapie, Band lx, p. 423–424.
Müller, Friedrich. Sammlung klinischer Vorträge, innere Medizin, 1905, No. 118.
Nothnagel. Topische Diagnostik. Centralwindungen, pp. 404–421.
Pierret, quoted by Bastian. Paralysis, Cerebral and Spinal, p. 284.
Starr. Brain Surgery, pp. 18–113.
Starr and McCosh. Amer. Jour. Med. Sci., 1894.
Spiller. Separate Sensory Centres in the Parietal Lobe, Jour. Nerv. and Ment. Dis., February, 1906.
Raynaud. Bull. d. l. Soc. anat., 1876, p. 522.
Rosenthal. Wien. med. Jahrb., April 26, 1878.
Russel and J. Horsley. Brain, 1906, Part cxlii, p. 137.
Walton and Paul. Clinical Value of Astereognosis, Jour. Nerv. and Ment. Dis., April, 1901.

A CONTRIBUTION TO THE PATHOLOGY OF REFRIGERATION FACIAL PALSY.

By L. Pierce Clark, M.D.,

VISITING NEUROLOGIST TO THE RANDALL'S ISLAND HOSPITALS AND SCHOOLS; CONSULTING NEUROLOGIST TO THE MANHATTAN STATE HOSPITAL, NEW YORK, AND THE CRAIG COLONY FOR EPILEPTICS, SONYEA, NEW YORK; ASSISTANT NEUROLOGIST TO THE VANDERBILT CLINIC (COLUMBIA UNIVERSITY), NEW YORK.

In order that the pathogenesis of Bell's palsy may be definitely determined we must insist that the type of cases studied shall be uniform in clinical occurrence and symptoms. Bell's palsy *per se* is a refrigeration palsy occurring in individuals who have no other organic disease. This statement, however, must not rule out such chronic predisposing diseases as rheumatism. In the refrigeration Bell's palsy a rheumatic history may or may not be present; it is much oftener absent. In the Vanderbilt Clinic statistics it was ten times more frequently absent than present.

The cause of the great frequency of paralysis of the facial nerve, as in Bell's type of palsy, is still moot. It is, however, due probably to anatomical causes. The nerve has a tortuous and exposed course through the Fallopian canal. It is easy to understand that inflammation may readily extend from the middle ear, or that cold may operate through the same channel, causing a local neuritis and palsy. In further substantiation of the vulnerability of the facial nerve to exposure, we know the facial nerve is infrequently paralyzed when the face is properly protected by hair or clothing. Bell's palsy is very infrequently seen in animals. Furthermore, it is reasonable to suppose that a certain number of individuals suffer a congenital narrowing of the foramen or canal through